Osteomyelitis of the Craniofacial Skeleton

David J. Pincus, M.D.,[1] Milton B. Armstrong, M.D.,[1] and Seth R. Thaller, M.D., D.M.D., F.A.C.S., F.A.A.P.[1]

ABSTRACT

Skull-based osteomyelitis, which is a true bony infection, originates from a chronic, inadequately treated infection. Because of the complex craniofacial skeletal anatomy and associated aesthetic concerns, osteomyelitis of the craniofacial skeleton must be uniquely managed and is more difficult to treat than osteomyelitis of other bones of the body. It is thought that osteomyelitis is decreasing in prevalence due to broad-spectrum antibiotic treatment; however, it still remains a challenging clinical entity in developing countries and lower socioeconomic areas.

KEYWORDS: Osteomyelitis, craniofacial infection, head and neck, skull base, osteomyelitis of the mandible

Skull-based osteomyelitis, which is a true bony infection, originates from a chronic infection, which has been inadequately treated. Because of the bony-based architecture and associated aesthetic concerns, osteomyelitis of the craniofacial skeleton must be managed differently than osteomyelitis of other bones of the body. This makes craniofacial infections more challenging to treat.[1] Because of widespread use of broad-spectrum antibiotics, it would be thought that osteomyelitis is decreasing in occurrence; however, it still remains a significant problem in developing countries and lower socioeconomic areas.[1] Osteomyelitis was first described in 1852 by a French surgeon, Edouard Chassaignac.[2] In 1764, John Hunter described pockets of dead cortical bone with abscess, which he termed sequestra.[2] He also described involucrum, or new bone formed in response to the sequestra.[2]

Pott, in 1778, postulated that osteomyelitis of the skull was caused by a bone contusion and extradural hemorrhage.[3] However, it was not until 100 years later that Van Launelongue classified cases into two types, primary or hematogenous osteomyelitis and secondary or contiguous osteomyelitis.[3] Currently, clinicians recognize that osteomyelitis of the skull is a complex disease with many different etiologies that requires prompt and definitive treatment.

Osteomyelitis is defined as an inflammatory condition of the bone that commences as an infection of the medullary cavity, rapidly involving the Haversian systems, and eventually involving the periosteum of the infected areas.[1] Invasion of bacteria into the cancellous bone results in compression of the blood vessels secondary to inflammation and edema of the marrow space. Severe compromise of the blood supply results in the development of ischemic and necrotic bone.[4] Immobility of the stagnant blood serves as a critical nidus for development of infection.[4] Etiology may result from trauma, bone surgery, bacteremia, or a contiguous infectious focus and is further influenced by diseases that affect the vascularity of bone, as well as by systemic diseases that produce an alteration of host defenses.[1] Examples of systemic diseases that decrease host defenses include diabetes, anemia, and malnutrition. Radiation, malignancy, osteoporosis, osteopetrosis, and

Paget's disease are all conditions that decrease the vascularity of bone and therefore cause a predisposition to infection.[1]

Anatomically, the bones involved in osteomyelitis of the skull include the mandible, frontal bone, maxilla, nasal bone, temporal bone, and skull base bones.[1] Although the diagnosis is quickly made clinically, radiologic imaging can be used for early detection and confirmation when the diagnosis is uncertain or to help gauge the severity of infection and extent of involvement.[1] Clinical sequelae of osteomyelitis include a draining sinus, periosteal thickening and tenderness, and bony destruction, with or without pathologic fractures confirmed by imaging studies. Consequences of such an infection can be as minor as a draining tract up to malignant transformation at the infected site.[1]

Functional imaging of the craniofacial skeleton is necessary for a complete treatment regimen in patients with osteomyelitis.[5] Computed tomography (CT) and magnetic resonance imaging (MRI) can be used for early detection.[1] Bone scintigraphy is more accurate than CT scan when used in detection of craniofacial osteomyelitis.[5] CT scans may show bone erosion or remodeling that may be confused with osteomyelitis.[5] The ability to view bone scans three-dimensionally due to the development of single photon emission computed tomography (SPECT) allows for precise anatomic delineation of bone activity, especially when the functional images of SPECT are fused with the structural images of high-resolution CT.[5]

Osteomyelitis is an opportunistic infection that is usually a complication of some other condition rendering the host susceptible to disease.[6] In tooth-bearing bone, osteomyelitis is usually caused by polymicrobial odontogenic bacteria. In long bones, classically *Staphylococcus aureus* is the offending microbe. *Bacteroides*, *Peptostreptococcus*, and microaerophilic *Streptococcus* spp., as well as other opportunistic pathogens, make up the odontogenic species that affect tooth-bearing bone.[6] Other newer-found organisms include *Arachnia*, *Klebsiella*, mycobacterium tuberculosis, and *Eikenella* spp. Fungal organisms, such as *Candida parapsilosis* and *Aspergillus*, also have been reported causing craniofacial osteomyelitis especially in patients who are immunocompromised. These organisms are probably derived from the original infections that were not properly treated either surgically or medically. This causes a proliferation of the original infection.[6]

Acute osteomyelitis may present as a routine infection with signs including fever, malaise, pain, and facial cellulitis.[6] There may not be any associated noticeable radiographic changes. It may take up to 10 to 12 days for bone loss to be apparent radiographically.[4] Acute osteomyelitis may be primarily managed with antibiotics. Also, underlying predisposing factors or conditions must be adequately addressed and treated. The antibiotic of choice is clindamycin because of its effectiveness against streptococci and the anaerobes that are usually found with osteomyelitis.[4] Hospitalization may be necessary for treatment with intravenous antibiotics. Surgical treatment is usually focused on debridement of involved soft tissue and bone. Where secondary to a mandibular or maxillary fracture, any nonviable teeth must be removed, as well as any loose bony fragments. Any foreign bodies (i.e., wires, plating) that may have been used for stabilization must also be removed.[4] The jaw must be subsequently restabilized with tight intermaxillary fixation or other choice of fixation technique (Fig. 1).

The figure below depicts a patient with frontal osteomyelitis as a complication of bacterial sinusitis. This complication of sinusitis is exceptionally rare in the antibiotic era. The main clinical findings include headache, sometimes associated with edema and spontaneous drainage if a sinocutaneous fistula has formed.[7] Early antibiotic treatment is administered for a minimum of 6 weeks. In some cases, especially when antibiotic therapy is ineffective, extensive bony debridement along with cranioplasty and placement of prosthesis may be necessary.[7]

In the above patient, cultures from middle ear fluid grew *Candida* and *Aspergillus*.[8] Fungal osteomyelitis is usually seen in immunocompromised patients. This patient in particular was an elderly man with diabetes
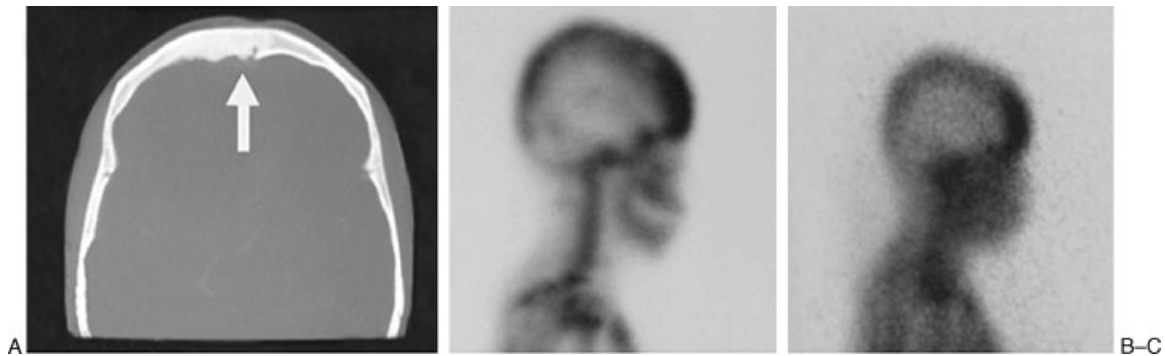

Figure 1   (A) A CT scan with frontal sinus inflammation. (B) Scintigraphy with inflammatory activity and osteomyelitis (^{99m}Tc). (C) Scintigraphy with inflammatory activity and osteomyelitis (^{67}Ga).

who presented with hearing loss and tinnitus for 2 months.[8] After a 4-week treatment with intravenous amphotericin B and an extensive oral course of itraconazole, the patient reported improvement of symptoms. Repeat imaging after 8 weeks showed residual mastoiditis without extension of disease.[8]

Chronic osteomyelitis may present like acute osteomyelitis with the inclusion of a chronic draining fistula.[6] Chronic osteomyelitis is a very serious condition requiring immediate hospitalization followed by aggressive surgical and antibiotic therapy.[4] In this disease, the blood supply to the infected area is usually severely compromised. At the time of surgery, culture material should be obtained so that an appropriate antibiotic can be chosen for treatment of the infection. Most authors agree that antibiotics should be continued much longer than usual for odontogenic infections.[4] For chronic osteomyelitis, antibiotic treatment may be continued for up to 6 months.[4]

As previously stated, osteomyelitis may be classified into acute and chronic forms. The former may be further divided into suppurative or nonsuppurative and then subclassified further. Acute forms of the disease may be caused by a contiguous focus such as trauma, surgery, or odontogenic infection. Other subclassifications include progressive or hematogenous (metastatic). Progressive forms may be a result of burns, sinusitis, or vascular insufficiency. Lastly, the hematogenous focus results from an infection that is introduced into the bloodstream and may arise from the developing skeleton or the developing dentition in children. Chronic osteomyelitis is also classified by causative agent. There may be a recurrent multifocal focus from the developing skeleton in children or from escalated osteogenic activity. Garré's osteomyelitis, a unique proliferative subperiosteal reaction in the developing skeleton, is another form of chronic infection. Suppurative or nonsuppurative as well as sclerosing forms may also be classified as chronic osteomyelitis. Sclerosing osteomyelitis may be broken down into diffuse and focal. Diffuse forms are caused by fastidious microorganisms secondary to compromised host and pathogen interface. Focal forms are predominately odontogenic caused by chronic localized injury.[6]

When classifying osteomyelitis, clinicians must consider the underlying condition of the host, the functional impairment caused by the disease, site of involvement, and extent of bony necrosis.[9] Cierny et al developed an anatomic classification system based on these criteria.

Cierny-Mader anatomic types of adult osteomyelitis are defined as follows: (A) Type 1 is intramedullary osteomyelitis, where the nidus is endosteal.[2] These do not necessarily require bone grafting.[9] (B) Type 2 indicates superficial osteomyelitis, which is limited to the surface of the bone.[2] Treatment involves hardware removal and debridement of avascular outer cortex.

Debridement is progressed down to the bleeding bone. This is called the paprika sign.[9] (C) Type 3 is termed localized osteomyelitis, in which the full thickness of the cortex of the bone is involved. This type of osteomyelitis often requires complex dead-space management and osseous stabilization after debridement.[2] This includes debridement, antibiotic beads, coverage, and bone grafting.[2] (D) Type 4 is diffuse osteomyelitis involving the entire circumference of the bone. These lesions are mechanically unstable and require complex reconstruction.[2] All infected or necrotic bone and soft tissue must be debrided. Preoperative blood supply must be sustained for successful treatment.[10] Dead space caused by extensive debridement must be managed appropriately. Some of the techniques include healing by secondary intention, closed irrigation and suction systems, temporary antibiotic polymethylmethacrylate (PMMA) beads, autologous bone graft, or vascularized bone grafts.[10] Defects in soft tissues can be treated by skin grafts, local muscle flaps, or vascularized free flaps.

It is important to obtain blood cultures in all patients with osteomyelitis, but it is especially important in those patients where hematologic spread is a concern.[6] Other useful laboratory values include elevated white blood cell count, certainly in the acute stages. Elevated erythrocyte sedimentation rate (ESR) and elevated C-reactive protein (CRP) may also be useful markers in the diagnosis and treatment of osteomyelitis.[6] ESR is not always reliable because fever, dehydration, and ongoing treatment with antibiotics may cause an elevation.[6] CRP assays may be used as a marker to monitor resolution of the infection in response to antibiotics or surgery.[6]

The treatment goal is directed to resolution of the infection while maximizing patient function. Celcius, in the 1st century AD, described scraping away or debridement of the dead bone until it bleeds.[2] Most patients with open fractures subsequently died before elucidation of the germ theory or understanding of the principles of infection. Early treatment of osteomyelitis of the long bones was subsequently amputation.[2] During World War I, Carel and Dakin used continuous irrigation to treat open fractures in soldiers.[2] After this period, Orr and Trueta recognized the importance of debridement of the sequestrum, stabilization of the bone, and maintaining the open wound.[2] These principles led to an increased success rate in the treatment of osteomyelitis, but still, 30% of patients had persistent local infection or systemic sepsis.[2]

Only in the past 25 years has the treatment of chronic osteomyelitis progressed to include use of muscle flaps and vascularized bone grafts to manage large open defects.[2] With use of new management techniques such as antibiotic beads to manage dead space in staged reconstructions, and use of external fixators in the Lizarov technique in the reconstruction of long bones, the success rate in the management of osteomyelitis is reported to be greater than 90%.[2]

As described by Rowlands et al, skull-based osteomyelitis secondary to pseudomonal infection presents in the elderly diabetic population as a severe unrelenting otitis externa progressing to the development of a unilateral facial nerve palsy, hearing loss, and progressively lower cranial nerve palsies.[10] Loss of the lower cranial nerves is also known as jugular foramen syndrome.[10] In this entity, the only manifestation of the skull-based osteomyelitis was the presentation of bilateral cranial nerve X palsies.[10] This is an atypical presentation of skull-based osteomyelitis that led to a delay in treatment and also highlights the limitations of CT imaging in the early stages of disease. The patient above did not demonstrate any further evidence of active otitis externa, middle ear disease, or sinus disease.[10]

Progressive bilateral cranial nerve palsies in elderly diabetic patients, even in the absence of an obvious concurrent focus of infection, should alert clinicians to the possibility of skull base osteomyelitis.[10] Jugular foramen syndrome is described by the development of multiple lower nerve palsies occurring when skull-based osteomyelitis involves the jugular foramen.[10] This usually indicates a poor prognosis. Although the infection in the patient above was caused by pseudomonal infection, *Aspergillus* infections have also been shown to cause skull-based osteomyelitis without any evidence of otitis externa.[10] *Aspergillus* infection usually presents as disease within the middle ear or mastoid, and in the reported cases none were diabetic.[10] The fact that CT scans showed no evidence of temporal bone involvement demonstrates the limitations of CT scan in the early stages of skull-based osteomyelitis.[10] This also highlights the usefulness of bony scans if there is any suspicion of osteomyelitis. ESR and CRP assays, as previously mentioned, are useful in this situation to monitor patient response to antibiotic therapy.[10]

Ng et al describe a case in which a 10-year-old immunocompetent patient developed a lateral medullary syndrome secondary to streptococcal milleri sphenoidal osteomyelitis.[11] Symptoms initially included headache and chronic sinusitis. As previously mentioned, skull-based osteomyelitis is usually caused by pseudomonal ear infections in elderly diabetic or immunocompromised patients.[10,11] Rarely, skull base osteomyelitis may be caused by paranasal infections.[11] Infectious organisms are usually *Aspergillus*, *Pseudomonas*, *Salmonella*, and *Staphylococcus* species.[11] Again, headache may be the only clinical symptom, with cranial neuropathies occurring later, thereby making early diagnosis difficult. In this case study, a 10-year-old immunocompetent girl developed lateral medullary syndrome (LMS) secondary to chronic sphenoid sinusitis.[11]

LMS, also known as Wallenberg syndrome, is a vascular syndrome occurring in the posterior circula-tion.[11] In order of frequency of occurrence, the neuro-logic sequelae include sensory deficits, dizziness/vertigo, and dysphagia.[11] Symptoms correspond with the level of medullary involvement. Headaches, dysphagia, and dys-arthria are more common in caudal lesions than in rostral lesions.[11] Although the mechanism of pathogenesis is unknown in these cases, it has been postulated that the symptoms are a result of direct extension of skull base osteomyelitis causing a thrombophlebitis vascular occlu-sion leading to a lateral medullary infarct.[11] This is in contrast with the thromboembolic event that is usually seen in LMS.

In these cases, an 8- to 12-week course of anti-biotics constitutes the main treatment strategy.[11] Endo-scopic sinus surgery has been increasingly described in recent years, but its role in the pediatric population remains controversial.[11] If there is continued clinical deterioration despite treatment, possible craniotomy with abscess drainage would be an option.[11]

MRI is superior to CT scan for early diagnosis of skull base osteomyelitis. MRI is more useful than CT for soft tissue discrimination and in assessing the soft tissue planes around the skull base and abnormalities of the medullary bone.[11] As previously mentioned, if CT or MRI results are inconclusive, then a bone scan with high inflammatory markers would be highly suggestive of skull base osteomyelitis.[11]

As described by Subburaman and Chaurasia, skull base osteomyelitis is a known complication of malignant external otitis (MEO).[12] MEO arises in elderly diabetic patients with an inflamed ear canal and granulations with or without facial weakness.[12] In some cases, in-volvement of the facial nerve and occasional radiologic findings of a mass, MEO can mimic malignancy.[12]

Several criteria have been described to distinguish between MEO and malignancy, including clinical fea-tures such as severe otalgia, granulation tissue in the external canal, isolation of *Pseudomonas aeruginosa*, and positive temporal bone scanning with technetium-99.[12] In malignant external otitis with skull base osteomyelitis, the infection travels via vascular and fascial planes.[12] It has been described that although there is dermal and osseous inflammation of the ear canal in MEO, the features are not specific.[12] Because of the age (68 years), facial nerve involvement, and the presence of soft tissue masses on CT and MRI, malignancy was suspected in the above patient.[12] After repeated unsuccessful naso-pharyngeal biopsies were performed, a gallium scan was performed, which led to establishing the diagnosis of skull-based osteomyelitis.[12] Subsequently, gallium scan-ning is useful in monitoring the effectiveness of treat-ment because the uptake reduces as the infection is controlled.[12] Scanning with technetium may still show positive results after the infection has been treated.[12] The facial nerve may not recover in these patients. Not only does the facial nerve get compressed by

granulations, but also it is affected by neurotoxins produced by *Pseudomonas aeruginosa*.[12] This illustrates the importance of medical treatment as opposed to surgical decompression. Appropriate treatment consists of long-term antibiotic therapy.[12]

Central skull base osteomyelitis has been described as occurring in the absence of otitis externa. Chang et al described a series of patients who presented with headache and cranial neuropathy without any external ear pain.[13] These atypical presentations arise from the sphenoid or occipital bones as opposed to the temporal bone seen in cases associated with otitis externa.[13] These cases occur less frequently and usually present solely with headache.[13] Gram-positive organisms are common with these infections, including underlying fungal infections of the sinuses with mucormycosis and *Aspergillus*.[13] This is in contrast with the *Pseudomonas* infections as seen in skull base osteomyelitis associated with otitis externa. In these atypical cases, imaging is best accomplished using MRI.[13] T1 hypointensity and T2 hyperintensity findings proved highly sensitive but nonspecific in the diagnosis of these cases, as described by Chang et al. It was noted that clival marrow and preclival soft tissue abnormalities were present in all these cases and were best appreciated on T1-weighted images.[13] Diagnosis was confirmed by tissue sampling using CT-guided fine-needle aspiration (FNA) of abnormal preclival tissue, sphenoidotomy, and open craniotomy.[13] In this series, elevated white blood cell count, fever, and abnormal blood cultures were absent. An elevated ESR level raised concern for skull base osteomyelitis and the need for tissue sampling.[13]

Several serious complications have been described in the literature occurring from skull base osteomyelitis, including cranial neuropathy, soft tissue involvement of the cavernous sinus with or without cavernous sinus thrombosis, and meningeal or brain parenchymal extension.[13] Cranial nerve involvement occurs due to the proximity of the clivus to the brain stem, basal cisterns, cavernous sinus, and skull-based foramina[13] (Fig. 2).
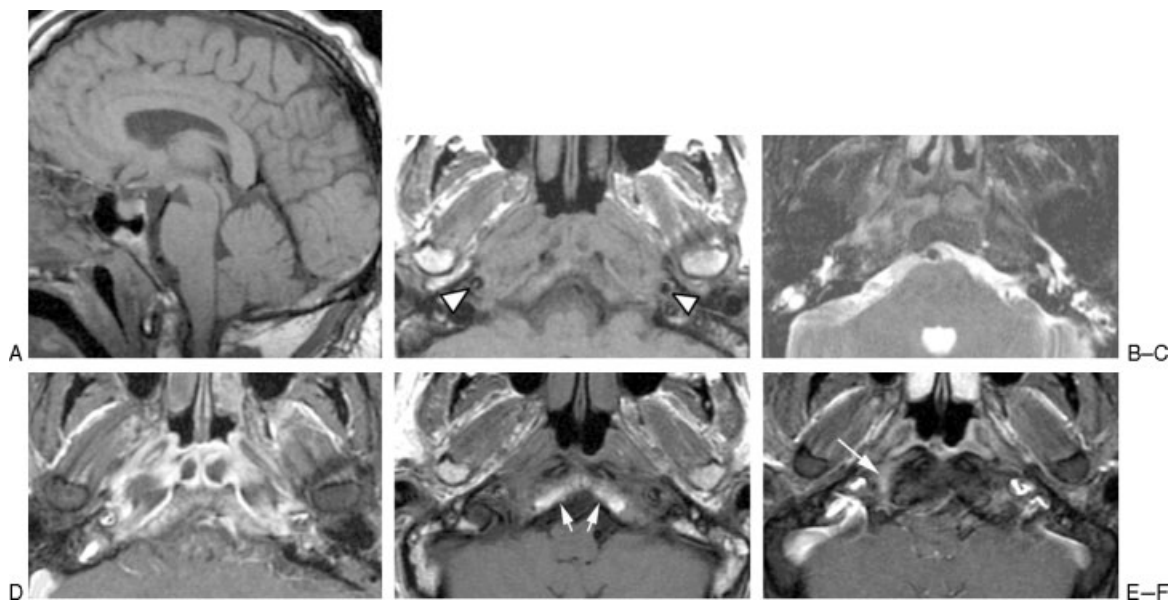

Figure 2 (A) Sagittal T1-weighted image demonstrates abnormally low signal intensity in the inferior clivus. (B) Axial T1-weighted image demonstrates abnormal soft tissue isointense to muscle infiltrating submucosally within the nasopharyngeal soft tissues, extending posteriorly to abut the carotid arteries (arrowheads), and replacing the normal hyperintense fatty marrow within the lower clivus. (C) Axial T2-weighted fast spin-echo image with fat saturation demonstrates mildly increased signal intensity within the infiltrated soft tissue compared with that of normal muscle. In addition, there is fluid in the mastoid air cells and middle ear cavities bilaterally, presumably related to Eustachian tube dysfunction or obstruction or both. (D) Contrast-enhanced axial T1-weighted spin-echo image with fat saturation demonstrates moderately intense enhancement of the infiltrative soft tissue. Some areas of nonenhancement may represent areas of infarcted or necrotic muscle. (E) Axial T1-weighted spin-echo image from a follow-up MR examination 16 months after the initial study demonstrates normalization of clival signal intensity (white arrows), as well as considerable reduction in bulk of the previously noted abnormal pre- and paraclival soft tissue. (F) Contrast-enhanced axial T1-weighted spin-echo image with fat saturation from the same follow-up examination demonstrates marked reduction of enhancement, with a normal appearance to the nasopharynx and prevertebral muscles. An ill-defined area of linear enhancement just at and anterior to the hypoglossal canal on the right (arrow) is likely extending along the course of the hypoglossal nerve. Of note, this patient's hypoglossal palsy has persisted despite resolution of all other symptoms. (Figure and description from Chang PC, Fischbein NJ, Holliday RA. Central skull base osteomyelitis in patients without otitis externa: imaging findings. AJNR Am J Neuroradiol 2003;24:1310–1316. Copyright © by American Society of Neuroradiology. Reprinted with permission.)

Singh et al described a series of patients with skull base osteomyelitis secondary to otitis externa who had been inadequately treated.[14] As a result, 66% of these patients died of the disease despite aggressive treatment, underlying the high morbidity and mortality associated with this disease.[14] Undertreatment remains the major contributor to this recurrence and contributes to the atypical features associated with this disease[14]; namely, unilateral severe otalgia, unremitting headache, elevated ESR, and unilateral malignant otitis externa. The recurrence rate is reported as 15 to 20%.[14] Other factors related to recurrence include failure to respond to antibiotics and infection by a nonpseudomonal organism in immunocompromised persons.[14] It is important to establish the signs and symptoms of recurrence so as to be diagnosed early and treated properly. These minor signs and symptoms include unremitting or persistent headaches in previously treated malignant otitis externa, otitis media with effusion in the ipsilateral or contralateral ear in the absence of nasopharyngeal lesions, and the presence of lower cranial nerve deficits especially in elderly diabetic individuals.[14]

Recurrence of otitis externa with associated skull base osteomyelitis has a high morbidity and mortality as stated earlier. Lower cranial nerve involvement depicts a poor prognosis despite aggressive antibiotic treatment. These patients also have a much higher morbidity.[14]

Chronic osteomyelitis of the jaw, also known as sclerosing osteomyelitis, has also been described in the literature.[15] Although its etiology remains unknown, this process occurs more commonly in females of any age group.[15] Symptoms include recurrent pain and swelling of the soft tissues overlying the involved mandible and enlargement and deformity of the hemi-mandible.[15] On imaging studies, patchy areas of sclerosis and radiolucency with thickening of the overlying muscle and subcutaneous tissues were seen.[15] The clinical course is described as chronic pain and inflammation with periods of exacerbation. It is deemed noninfectious in origin due to the lack of positive microbiological cultures.[15] Antibiotic treatment, nonsteroidal anti-inflammatory drugs, and steroid drugs seem to control symptoms of these patients; however, these do not seem to alter the frequency of recurrence.[15] Surgical intervention, including decortication, does have a role in these patients. Resection may be required in severe cases to achieve adequate margins and removal of devitalized teeth.[15]

For reconstruction of significant defects of the head and neck, free tissue transfer has become the treatment of choice.[16] The literature states that the success rate of these free flaps for reconstruction approaches 95%.[17] Donor flap sites include the radial forearm, fibula, iliac crest, rectus, and latissimus dorsi.[17] Optimum results are achieved when performing face tissue transfer using donor sites with large-caliber vessels, the use of vein grafts, imaging studies, physical examination, and patient history to evaluate successful treatment.[17] Gbara et al described a series of patients who underwent free fibula flap for reconstruction of the maxilla and mandible.[17] Many times these osseous defects are large causing functional and aesthetic deficits, necessitating a reconstructive procedure.[18] Methods of harvesting the flap and the bone include free bone transplantation using cortico-cancellous bone, titanium mesh with cancellous bone chips, and vascularized bone grafts.[18] Vascularized flaps, providing their own blood supply, may be free or pedicled allowing for transposition. Free flaps require reanastomosing at the recipient site, but the biological advantage of pedicle flaps are the resistance to infection and increased wound healing.[18] The literature states that fibula flaps are very stable due to their high cortical content and the high mechanical rigidity of cortical bone, as well as their high protein content, which promotes the healing process.[18] These grafts are usually stabilized prosthetically by means of a supportive bar that is screwed onto the implant or by magnetic inserts.[18]

Valentini et al described a series of patients with extensive craniofacial bony defects using autogenous bone grafts.[18] As stated previously, donor sites included ilium, rib, mandibular symphysis, maxillary shaft, retromolar area, calvaria, and scapula.[19] The literature refers to autogenous bone as the material of choice due to its potential for revascularization and its osteoconductive and osteoinductive properties.[19]

According to Gilles' principle, any defect should be restored with local flaps when possible. Both aesthetic and functional outcomes are nearly always superior to any graft or distant flap.[19] Depending on the flaps needed, different surgical approaches are necessary. There are similarities in technique to help ensure a successful outcome. Schusterman et al describe use of the AO reconstruction plate for a fixation device in mandibular reconstruction cases using vascularized bone flaps.[20] Advantages to this device is that it can be readily molded into any type of mandibular defect. The device remains strong enough to maintain the mandible in rigid fixation.[20] The literature states that these plates are useful for lateral mandibular fractures, especially when used with some type of flap, but inadequate for most cases necessitating an anterior mandibular reconstruction. In these cases, the free vascularized bone transfer was the technique of choice.[20]

Lastly, it is important to discuss orbit and midface reconstruction using osseous free flaps. Structures of the midface include the maxilla, paranasal sinuses, inferior orbits and zygomas. Osseous pedicled flaps that consist of vascularized cranial bone have been described in the literature, but these can only be used for small defects.[21] Although the use of maxillofacial prosthesis for reconstruction is unavoidable in midface repair, osseous free flaps can assist in the overall management of these

patients.[21] A drawback with prosthetic devices is that they cannot be used when there is insufficient bony support. In these cases, osseous free flaps can be used in conjunction with a prosthetic device to help improve contour and provide further bony support to the orbit and midface.[21]

Osteomyelitis of the craniofacial skeleton is a complex problem requiring rapid and thorough diagnosis and treatment. Failure to do so can result in a host of complications and consequences. The cause of this disease is multifactorial and its presentation varies. Whatever the cause may be, complete resolution of the infection must be obtained to decrease the morbidity and mortality of the patient.

REFERENCES

1. Prasad KC, Prasad SC, Mouli N, Agarwal S. Osteomyelitis in the head and neck. Acta Otolaryngol 2007;127:194–205
2. Flemming H. Osteomyelits of the skull. California and Western Medicine 1925;XXIII:985–988
3. Osteomyelitis. In: Peterson L, ed. Contemporary Oral Maxillofacial Surgery. 4th ed. Philadelphia,PA: Mosby Publishers; 2003:375–377
4. Strumas N, Antonyshyn O, Caldwell CB, Mainprize J. Multimodality imaging for precise localization of craniofacial osteomyelitis. J Craniofac Surg 2003;14:215–219
5. Wallace-Hudson J. Osteomyelitis and osteoradionecrosis. In: Fonseca RJ, ed. Oral and Maxillofacial Surgery: Surgical Pathology. Philadelphia, PA: WB Saunders; 2000:484–490
6. Tuon FF, Russo R, Nicodemo AC. Brain Abscess Secondary to Frontal Osteomyelitis. Revista do Instituto de Medicina Tropical de Sao Paulo. 2006; Available at: www.scielo.br. Accessed August 27, 2007
7. Naeem A, et al. Fungal osteomyelitis of the skull base. Infect Med 2003;20:535
8. Cierny G, Mader JT, Penninck JJ. A clinical staging system for osteomyelitis. Clin Orthop Relat Res 2003;414:7–24
9. Parsons B, Strauss E. Surgical management of chronic osteomyelitis. Am J Surg 2004;188:57–66
10. Rowlands RG, Lekakis GK, Hinton AE. Masked pseudomonal skull base osteomyelitis presenting with a bilateral Xth cranial nerve palsy. J Laryngol Otol 2002;116:556–558
11. Ng J, Connolly DJ, Rittey CD, Mordekar SR. Skull base osteomyelitis leading to lateral meduallry syndrome in a child. Eur J Paediatr Neurol 2007;11:111–114
12. Subburaman N, Chaurasia MK. Skull base osteomyelitis interpreted as malignancy. J Laryngol Otol 1999;113:775–778
13. Chang PC, Fischbein NJ, Holliday RA. Central skull base osteomyelitis in patients without otitis externa: imaging findings. AJNR Am J Neuroradiol 2003;24:1310–1316
14. Singh A, Al Khabori M, Hyder MJ. Skull base osteomyelitis: diagnositic and therapeutic challenges in atypical presentation. Otolaryngol Head Neck Surg 2005;133:121–125
15. Heggie AA, Shand JM, Aldred MJ, Talacko AA. Juvenile mandibular chronic osteomyelitis: a distinct clinical entity. Int J Oral Maxillofac Surg 2003;32:459–468
16. Varvares MA, Lin D, Hadlock T, et al. Success of multiple, sequential, free tissue transfers to the head and neck. Laryngoscope 2005;115:101–104
17. Gbara A, Darwich K, Li L, Schmelzle R, Blake F. Long term results of jaw reconstruction with microsurgical fibula grafts and dental implants. J Oral Maxillofac Surg 2007;65:1005–1009
18. Valentini V, Cassoni A, Marianetti TM, et al. Reconstruction of craniofacial bony defects using autogenous bone grafts: a retrospective study on 233 patients. J Craniofac Surg 2007;18:953–958
19. Tan O, Atik B, Ergen D. Temporal flap variations for craniofacial reconstruction. Plast Reconstr Surg 2007;119:152e–163e
20. Schusterman MA, Reece GP, Kroll SS, Weldon ME. Use of the AO plate for immediate mandibular reconstruction in cancer patients. Plast Reconstr Surg 1991;88:588–593
21. Schusterman MA, Reece GP, Miller MJ. Osseous free flaps for orbit and midface reconstruction. Am J Surg 1993;166:341–345